Taiwanese Journal of Obstetrics & Gynecology 56 (2017) 425–431

Contents lists available at ScienceDirect

Taiwanese Journal of Obstetrics & Gynecology

journal homepage: www.tjog-online.com

Review Article

Prenatal diagnosis of congenital lobar fluid overload

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ARTICLE INFO

Article history:
Accepted 3 May 2017

Keywords:
Bronchopulmonary sequestration
Congenital lobar fluid overload
Congenital pulmonary airway malformation
Fetus
MRI

ABSTRACT

Prenatal congenital lobar fluid overload (CLFO), which was first described by Ramsay and Byron, is identical to postnatal congenital lobar overinflation. It is characterized by progressive lobar overexpansion that compresses the other adjacent lung lobes. The underlying cause can be an intrinsic cartilaginous abnormality or an extrinsic airway compression. It may be associated with cardiovascular anomalies in 12%–14% of cases and affects males more frequently than females. Most cases are diagnosed postnatally, but early antenatal diagnosis and sequential follow-up are attempted for early treatment, if clinically indicated. This article provided a thorough review of CLFO, including prenatal diagnosis and differential diagnoses, as well as comprehensive illustrations of the perinatal imaging findings of CLFO. Prenatal diagnosis of fetal lung lesions should include CLFO in the differential diagnosis and prompt investigation for associated anomalies.

Introduction

Congenital lobar fluid overload (CLFO) in fetuses, which was first described by Ramsay and Byron and is also referred to as congenital lobar overinflation (CLO) or congenital lobar emphysema (CLE) postnatally, is a rare cystic lung lesion that can often be diagnosed accurately in the neonatal or infantile periods based on presentations of respiratory distress and hyperinflation of pulmonary lobes [1,2]. CLFO is a disease entity of fluid-overloaded and expanded lung tissue, which usually presents on antenatal ultrasonography (US) as a homogeneous hyperechogenicity with or without mass effect to the mediastinum [1,3–19]. On magnetic resonance imaging (MRI), hyperintensity of the affected lung lobe, mass effect to the mediastinum, and intact lung architecture with stretched hilar vessels were frequently observed [1,5,6,11–13,20–27]. From our experience, relative hypointensity of the adjacent and compressed lung lobe is common (Figs. 1–6, cases 1–3), but hyperintensity of the compressed lung tissue can be seen in some cases [11]. The etiology of this malformation remains unclear, but several theories were proposed; these include 1) a bronchial cartilaginous defect that produces a one-way valve effect; 2) endobronchial mucus impaction or proliferative mucosal infolding (polyalveolosis or polyalveolar lobe); 3) extrinsic compression of the bronchi from aberrant vessels or lung parenchymal lesions; and 4) atresia of a lobar bronchus [28–30]. The common locations of CLFO, in the order of frequency, are as follows: left upper lobe, right middle lobe, and right upper lobe [4,31]. Males are more frequently affected than females [32] and about 20% of patients with CLFO have associated congenital cardiovascular anomalies [33,34].

Prenatal diagnosis

Fetal CLFO is usually discovered in the second trimester. Up to the present time, at least 41 cases of fetal CLFO have been

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http://dx.doi.org/10.1016/j.tjog.2017.05.001

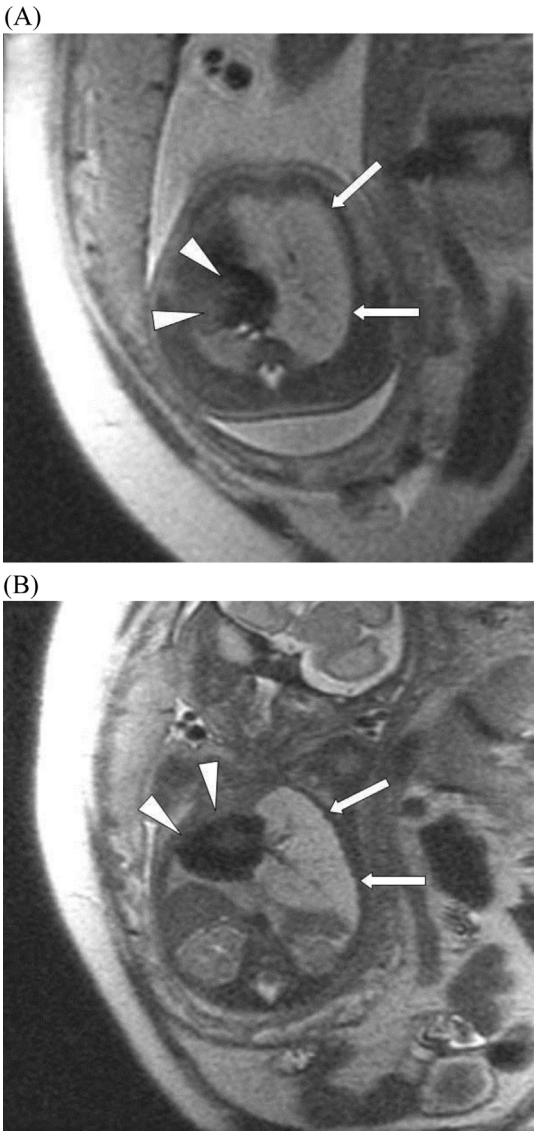

Fig. 1. (A) Axial and (B) coronal MRI of CLFO in case 1 at 20 weeks gestational age shows hyperintense and expanded left upper lobe (arrows), suggesting fluid overload and causing compression of the heart toward the right (arrowheads).

reported in English literature (Table 1). Quinton et al. have reported the earliest diagnosis of CLFO at 18 gestational weeks [4]. Prenatal US remains the reference standard of imaging modalities to evaluate the lesion. The most common manifestation on US is a unilateral, bright, hyperechogenic lesion in the affected lung [5], but Alamo et al. [13] and Babu et al. [17] reported that some CLFO cases can present as cystic mass lesions (Table 1). On fetal MRI, CLFO appears as a fluid-overloaded segment or lobe with high signal intensity and stretching of the hilar vessels without architectural distortion of the adjacent pulmonary lobes [1]. Some cases show partial or complete regression on serial follow-up US in the third trimester, depending of the etiology of the blockage. However, it is sometimes difficult to distinguish CLFO from microcystic congenital pulmonary airway malformation (CPAM) and bronchopulmonary sequestration (BPS) on prenatal US [1]. Nowadays, fetal MRI can be a helpful complementary modality to US to differentiate among CLFO and other fetal cystic lung lesions owing to its better tissue contrast, larger field of view, enhanced anatomic evaluation, more detailed

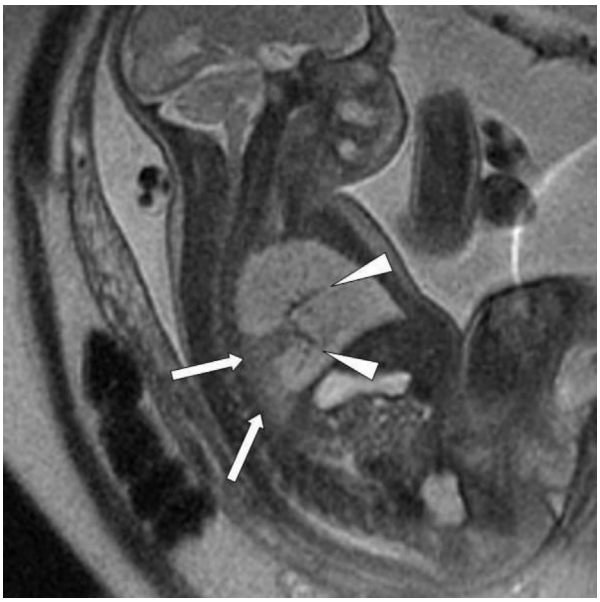

Fig. 2. Sagittal MRI of CLFO in case 1 at 20 weeks gestational age shows a hyperintense and expanded left upper lobe and a small, hypointense, compressed adjacent left lower lobe (arrows). Intact lung architecture with stretched hilar vessels (arrowheads) is observed. The lesion disappeared at 37 weeks gestational age and postnatal chest radiograph of the infant was normal.

demonstration of the lesion extent, as well as the capability of detecting other associated congenital anomalies.

Differential diagnosis

Fetal cystic lung lesions are rare but significant in the spectrum of congenital lung malformations (CLMs). These lesions include CLFO, which is synonymous with postnatal CLE, BPS, CPAM, and bronchial atresia (BA) [1]. Antenatal differential diagnoses of CLFO should be done for appropriate treatment recommendations.

Congenital pulmonary airway malformation

CPAM, previously known as congenital cystic adenomatoid malformation (CCAM), is characterized by adenomatoid proliferation of bronchiole-like cysts in the lung and lack of normal alveolar development [35]. It has been the most commonly diagnosed lung malformation prenatally, accounting for 30%–40% of all congenital lung diseases [20]. The outcome of CPAM is unpredictable. Some postulate that CPAM with <57% of the total lung volume would resolve completely, whereas CPAM with >84% of total lung volume will not [1]. One important prognostic factor is the presence of hydrops; if not treated, more than 90% of these fetuses die before birth [36]. CPAM is thought to be a predisposing condition to lung neoplasms, such as pleuropulmonary blastoma or rhabdomyosarcoma [5]. Malignant transformation to bronchoalveolar carcinoma is another issue that was reported to develop on a preexisting CPAM in young adulthood [5].

Stocker et al. distinguished three types of CPAM based on cyst size and histopathology. Type I consisted of large cysts (at least one dominant cyst >1 cm in diameter); type II comprised numerous small cysts <1 cm; and type III has microcystic lesions <0.2 cm in diameter [37]. Nowadays, this classification had been expanded to include two additional subtypes: type 0 displays acinar dysgenesis or dysplasia, whereas type IV exhibits distal acinar origin [37].

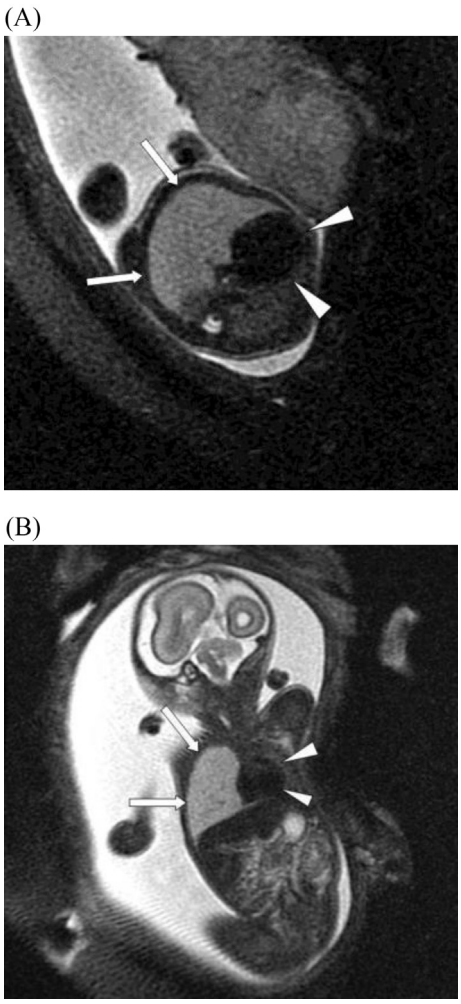

Fig. 3. (A) Axial and (B) coronal MRI of CLFO in case 2 at 23 weeks gestational shows hyperintense and expanded right upper lobe (arrows), suggesting fluid overload and causing compression of the heart toward the left (arrowheads).

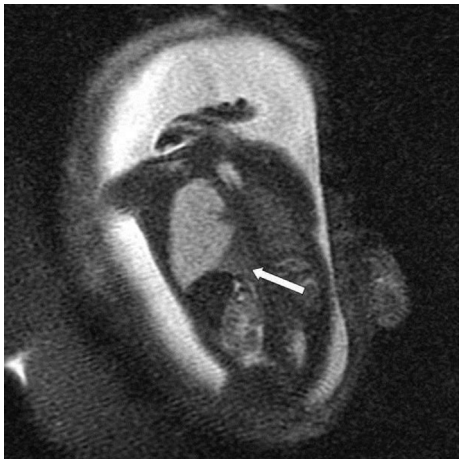

Fig. 4. Coronal MRI of CLFO in case 2 at 23 weeks gestational shows hyperintense and expanded right upper lobe and hypointense small compressed right lower lobe (arrow). The diagnosis of CLE was confirmed by lobectomy after birth due to neonatal respiratory distress.

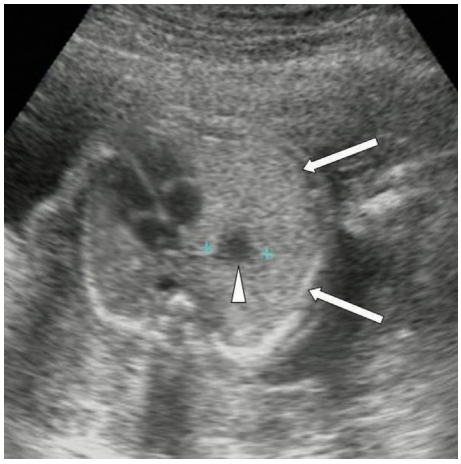

Fig. 5. Prenatal ultrasound of a CLFO that presents as a parahilar cystic lesion in case 3. Chest US at 21 weeks gestational age reveals a homogeneous hyperechogenicity of the left lung (arrows) shifting of the heart to the right. A small nodule with hypoechogenicity over the left parahilar region is found (arrowhead).

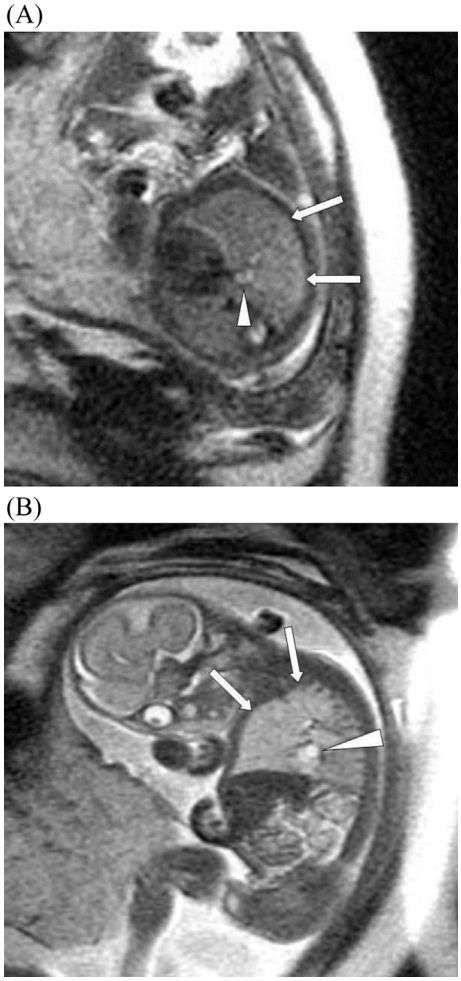

Fig. 6. (A) Axial and (B) sagittal MRI of a CLFO that presents as a parahilar cystic lesion in case 3 at 22 weeks gestational age shows hyperintense and expanded left upper lobe (arrows), suggesting fluid overload and causing compression of the heart toward the right. A small bright nodule over the left parahilar region is found (arrowhead). Bronchogenic cyst was proven postnatally.

Table 1
Ultrasound and/or magnetic resonance imaging (MRI) findings in previous reported cases of fetal congenital lobar fluid overload.

Authors [Reference]	Location	GW at prenatal sonographic diagnosis	Prenatal sonographic findings	GW at prenatal MRI diagnosis	Prenatal MRI findings	Perinatal outcome
Liu et al. [1]						
Case 1	Left upper lobe	29	Microcystic CCAM appearance	30	CLFO of LUL with compression of LLL inferiorly	CLE due to bronchial cartilage defect histologically
Case 2	Left upper lobe	25	Microcystic CCAM appearance	25	CLFO of LUL with compression of LLL inferiorly	Lesion resolution or disappearance on follow-up US
Case 3	Right upper lobe	22	Microcystic CCAM with a right hilar cyst	22	Macrocystic CCAM in the right parahilar region with RUL CLFO pattern	Resolution of CLFO due to regression of macrocystic CCAM on follow-up US; histologically confirmed CLE due to a macrocystic CCAM
Quinton et al. [4]	Right middle and lower lobes	18	Enlarged uniformly echogenic right lung with mediastinal shift and left lung compression			Lesion disappeared in 29 + 5 GW; Histology revealed CLE with deficient bronchial cartilage
Lacy et al. [8]						
Case 1		18	Unilateral bright echogenic lesion			No size diminution of the lesion on follow-up US; Lobectomy and histology revealed CLE (CMV infection may be one possible cause histologically)
Case 2		18	Unilateral bright echogenic lesion			No size diminution of the lesion on follow-up US; Lobectomy and histology revealed CLE
Chia et al. [9]	Left lung	Due date	A left lung tumor was suspected and the fetal heart has been pushed to the right side			Histology revealed CLE
Tobias et al. [10]	Left upper lobe	20	Microcystic echogenic mass			Histology revealed CLE
Oluyinka et al. [11]						
Case 1	Right middle lobe	20	A homogeneous echogenic mass with mediastinal shift and compression of the left lung.	20	A homogeneous hyperintense mass with mediastinal shift and compressed right lung	Lesion resolved in 36 GW; Histology revealed CLE
Case 2	Right lower lobe	23	A homogeneous echogenic mass with mediastinal shift	23	Subtle hyperintensity of the right lower lung	Slight resolution of size until delivery; Histology revealed bronchial dysplasia with CLE
Alamo et al. [13]						
Case 1			Homogeneous hyperechogenicity	22	Homogeneous T2-hyperintensity	Obvious radiologic sign of CLE postnatally
Case 2			Homogeneous hyperechogenicity	25	Homogeneous T2-hyperintensity	Histology revealed bronchial atresia combined with CLE
Case 3			Multiple small cysts	28	Homogeneous T2-hyperintensity	Obvious radiologic sign of CLE postnatally
Case 4			Homogeneous hyperechogenicity	25	Homogeneous T2-hyperintensity	Obvious radiologic sign of CLE postnatally
Case 5			Homogeneous hyperechogenicity	22	Homogeneous T2-hyperintensity	Obvious radiologic sign of CLE postnatally
Case 6			Homogeneous hyperechogenicity	25	Homogeneous T2-hyperintensity	Obvious radiologic sign of CLE postnatally
Case 7			Homogeneous hyperechogenicity	30	Homogeneous T2-hyperintensity	Obvious radiologic sign of CLE postnatally
Case 8			Homogeneous hyperechogenicity	31	Homogeneous T2-hyperintensity	Obvious radiologic sign of CLE postnatally
Richards et al. [14]	Right lung	24	Homogeneous echogenic mass with mediastinal shift; polyhydramnios			Lesion resolved in 37 GW; Histology revealed CLE
Okabe et al. [15]	Right middle lobe		Cystic lesion			Histology revealed CLE due to bronchiectasis

Table 1 (*continued*).

Authors [Reference]	Location	GW at prenatal sonographic diagnosis	Prenatal sonographic findings	GW at prenatal MRI diagnosis	Prenatal MRI findings	Perinatal outcome
Wansaicheong et al. [16]	Right lower lobe, anterior segment	22	Homogeneous echogenic mass with mediastinal shift			Lesion resolved in 33 GW; Obvious radiologic sign of CLE postnatally
Babu et al. [17]	Right upper and middle lobes	22	Microcystic and macrocystic echogenic mass with mediastinal shift			Histology revealed CLE
Carrol et al. [18]	Left upper lobe	29	Echogenic mass with mediastinal shift; polyhydramnios			Histology revealed CLE with mild interstitial inflammation and CMV inclusion bodies
Kasales et al. [19]	Right lung		Homogeneous mass with mediastinal shift			Lesion persists on follow-up US; Obvious radiologic sign of CLE postnatally
Pacharn et al. [23]						
Case 1	Left upper lobe			25	A homogeneous T2-hyperintense lesion in the left upper lobe	Obvious radiologic sign of CLE postnatally
Case 2	Right lower lobe			25	A small homogeneous T2-hyperintense area in the medial basal segment of the right lower lobe	Obvious radiologic sign of CLE postnatally
Case 3 and Case 4	*					Obvious radiologic sign of CLE postnatally
Cases 5–9	*					Histology revealed CLE
Truitt et al. [25]	Right middle lobe	20	Right-sided thoracic mass	22	Overinflated right middle lobe	The lesion gradually resolved on follow-up US; Histology revealed CLE
Kunisaki et al. [26]						
Case 1				30	A large left upper lobe mass	Obvious radiologic sign of CLE postnatally
Case 2	*					Obvious radiologic sign of CLE postnatally
Cases 3–5	*					Histology revealed CLE, all with cystic adenomatoid and polyalveolar changes
Takahiko et al. [27]						
Case 1	Left upper lobe	25	Massive enlargement of the left upper lobe	30	Large left upper lobe and solitary cyst at the hilum	Histology revealed CLE with small bronchial lumen
Case 2	Left upper lobe	26	Massive enlargement of the left upper lobe with mediastinal shift			The size of the left upper lobe gradually decreased during the pregnancy; Histology revealed CLE due to stenotic proximal bronchus with hypertrophied, fused bronchial cartilages

GW, gestational age.*, Not available.

Some authors simply differentiate cystic lung lesions antenatally detected by US into two types based on diameter: macrocysts (≥5 mm) and microcysts (<5 mm) [38].

These malformations may comprise cystic and solid components, resulting in various US and MRI manifestations [21]. On fetal US, CPAMs could be demonstrated as uniform hyperechogenic masses, anechoic cystic masses, or multicystic masses with echogenic stroma; most of these are confined to one segment or one lobe of the lung [37,39]. The typical MRI findings depend on the size of the cysts. The signal intensity of the cysts is higher than that of the surrounding normal lung parenchyma [1,22,40]. Macrocystic CPAM presents as a mass with lobulated margin and inhomogeneous hyperintensity [1,20,22], whereas microcystic CPAM is seen as a lobulated mass with homogeneous hyperintensity and arterial vascular architectural distortion without identifiable cysts [1,23]. Partial or complete regression of the masses *in utero* appears to be the rule. Compared to a normal lung, regressed CPAM usually appears as an ill-defined, non-homogeneous, and slightly hyperintense mass, which shows gradually decreasing signal intensity on follow-up MRI [1,22]. Fetuses with isolated CPAM have good prognoses regardless of the histologic type [24].

Bronchopulmonary sequestration

BPS comprises nonfunctioning pulmonary tissue that fails to connect with the normal tracheobronchial tree and receives arterial blood supply from the systemic circulation [1,22,25,41]. BPS accounts for about 23% of prenatally detected lung lesions [25] and is

the second most common lung lesion found on antenatal diagnosis [24]. The most frequent (more than two-thirds) location is the left lower lobe [42]. BPS is subdivided into two types based on the pleural covering. Intralobar sequestration lies within the normal lung parenchyma without its own pleura and drains into the pulmonary vein, whereas the extralobar type has its own distinct pleural covering with complete anatomic separation from the rest of the lung and drains to the systemic veins [5,23]. Sometimes, BPS had been thought to be a kind of systemic arteriovenous malformation that may cause cardiovascular symptoms or even lead to cardiac failure [3,5]. Large lesions can compress the esophagus and thoracic veins to cause fetal hydrops, which is an established indication for fetal intervention and early delivery [43]. The intralobar type is more common and is usually associated with type II CPAM (hybrid lesions), but is rarely diagnosed prenatally [44]. Most antenatally diagnosed lesions are of the extralobar type of sequestration, which may be associated with other congenital systemic anomalies, such as congenital diaphragmatic hernia or cardiac abnormalities [3]. More than 50% of lesions regress partially or completely *in utero* [1,45].

On fetal US, BPS present in most cases as hyperechogenic, homogeneous masses that are commonly located in the left lower lung, inferior to the lung, or even below the diaphragm [3]. Systemic arterial supply could be disclosed by color Doppler US [3]. Usually, prenatal US cannot distinguish between intralobar and extralobar BPS [45]. The MRI findings of a pure BPS include a well-defined, homogeneous, triangular hyperintense mass relative to the surrounding normal lung, but with less signal intensity than the amniotic fluid, as well as visualization of the systemic feeding artery [1,20,38,6]. In hybrid lesions of BPS and CPAM, an inhomogeneous hyperintense mass with lobulated contour could be observed [1,20,23]. Regressed BPS usually has a lobulated margin with decreased and inhomogeneous signal intensity [1,41]. The key point in differentiating BPS from CPAM type III and overinflation is demonstration of systemic arterial supply on both MRI and US [23].

Bronchial atresia

BA is a rare anomaly that is characterized by obliteration of a segmental or subsegmental bronchus near its origin, with normally developed distal structures and associated collateral alveolar fluid drift, which is different from fluid trapping in CLFO. The obstructed bronchus causes lung fluid accumulation with dilatation of the bronchi (i.e., bronchocele) and distal lung expansion [3]. It is most commonly seen in the apical and posterior segments of the left upper lobe. A strong association of BA with other congenital lung anomalies, such as CPAM, intralobar BPS, and CLFO, has been reported and raised the concern of a common embryonal origin [7,26]. Although the diagnosis of isolated BA is increasing recently, correct prenatal characterization remains a challenge.

On fetal US, the involved lung segment is seen to expand with homogeneous hyperechogenicity [3]. On fetal MRI, the involved lung is expanded and shows high signal intensity, with focal bronchial dilatation if the proximal bronchus is affected. In difficult cases of distal bronchial obliteration, only pulmonary expansion with signal intensity change could be seen on MRI [3]. If postnatal computed tomography is performed, both mucus-filled bronchus and adjacent air trapping could be seen.

Fetal outcome

Fetal prognosis depends on changes in the size of the mass, as well as the presence of polyhydramnios and hydrops fetalis. Most CLFO cases show partial or complete regression *in utero* during the third trimester. About 20% of patients with CLFO have associated congenital cardiac anomalies [33,34] that might cause fetal cardiopulmonary compromise. Two infant series (10 infants in one series and 6 in the other) disclosed no deaths in CLFO patients after surgical treatment [46,47] implying good prognosis and survival. Antenatal detection of CLFO allows serial follow-up during pregnancy and early management before the infant becomes symptomatic postnatally [4].

Conclusion

All patients with prenatally detected CLMs, like CLFO, require thorough postnatal evaluation, including chest computed tomography scan. Postnatally, the clinical appearance of CLMs can vary from immediate respiratory distress at birth to an incidental chest radiograph finding at any age [5]. Even if these CLMs seem to disappear on late trimester imaging with a normal postnatal chest radiograph, some authors recommend imaging follow-up to make sure that there are no residual subtle abnormalities [1] although cystic lung lesions can spontaneously decrease in size and lead to improved mediastinal shift [48], complete postnatal resolution is rare [49,50]. Imaging follow up is also important to determine when and how to investigate those infants [24,8]. US is the modality of choice for routine imaging of the fetus, but MRI had been recently shown to complement US. The advantages of fetal MRI include its large field of view and excellent soft tissue contrast. Compared with US, MRI is less affected by maternal habitus, fetal position, and level of amniotic fluid [24]. MRI is also useful in determining lung volume, which has a prognostic implication on postnatal pulmonary function, as well as in planning both *in utero* interventional procedures and immediate postnatal treatment. In the future, cooperation between gynecologists and radiologists, and correlation of prenatal US with MRI are beneficial and essential [22].

Conflicts of interest

The authors have no conflicts of interest relevant to this article.

Acknowledgement

The review study was approved by the Institutional Review Board of MacKay Memorial Hospital (IRB Number: 16MMHIS056).

References

[1] Liu YP, Chen CP, Shih SL, Chen YF, Yang FS, Chen SC. Fetal cystic lung lesions: evaluation with magnetic resonance imaging. Pediatr Pulmonol 2010;45:592–600.
[2] Ramsay BH, Byron FX. Mucocele, congenital bronchiectasis and bronchiogenic cyst. J Thorac Surg 1953;26:21–30.
[3] Alamo L, Gudinchet F, Reinberg O, Vial Y, Francini K, Osterheld MC, et al. Prenatal diagnosis of congenital lung malformations. Pediatr Radiol 2012;42:273–83.
[4] Quinton AE, Smoleniec JS. Congenital lobar emphysema-the disappearing chest mass: antenatal ultrasound appearance. Ultrasound Obstet Gynecol 2001;17:169–71.
[5] Eber E. Antenatal diagnosis of congenital thoracic malformations: early surgery, late surgery, or no surgery? Semin Respir Crit Care Med 2007;28:355–66.
[6] Biyyam DR, Chapman T, Ferguson MR, Deutsch G, Dighe MK. Congenital lung abnormalities: embryologic features, prenatal diagnosis, and postnatal radiologic-pathologic correlation. Radiographics 2010;30:1721–38.
[7] Langston C. New concepts in the pathology of congenital lung malformation. Semin Pediatr Surg 2003;12:17–37.
[8] Lacy DE, Shaw NJ, Pilling DW, Walkinshaw S. Outcome of congenital lung abnormalities detected antenatally. Acta Paediatr 1999;88:454–8.
[9] Chia CC, Huang SC, Liu MC, Se TY. Fetal congenital lobar emphysema. Taiwan J Obstet Gynecol 2007;46:73–6.
[10] Ankermann Tobias, Oppermann HC, Engler S, Leuschner I, Von Kaisenberg CS. Congenital masses of the lung, cystic adenomatoid malformation versus

congenital lobar emphysema: prenatal diagnosis and implications for postnatal treatment. J Ultrasound Med 2004;23:1379–84.
[11] Olutoye OO, Coleman BG, Hubbard AM, Adzick NS. Prenatal diagnosis and management of congenital lobar emphysema. J Pediatr Surg 2000;35:792–5.
[12] Epelman M, Kreiger PA, Servaes S, Victoria T, Hellinger JC. Current imaging of prenatally diagnosed congenital lung lesions. Semin Ultrasound CT MR 2010;31:141–57.
[13] Alamo L, Gudinchet F, Meuli R. Comparison of foetal US and MRI in the characterisation of congenital lung anomalies. Eur J Radiol 2013;82:860–6.
[14] Richards DS, Langham Jr MR, Dolson LH. Antenatal presentation of a child with congenital lobar emphysema. J Ultrasound Med 1992;11:165–8.
[15] Okabe K, Hara K, Ando A, Sano S, Shimizu N, Teramoto S, et al. Congenital lobar emphysema successfully treated by right upper lobectomy at five hours after delivery: a case report. Kyobu Geka 1992;45:590–4.
[16] Wansaicheong GK, Ong CL. Congenital lobar emphysema: antenatal diagnosis and follow up. Australas Radiol 1999;43:243–5.
[17] Babu R, Kyle P, Spicer RD. Prenatal sonographic features of congenital lobar emphysema. Fetal Diagn Ther 2001;16:200–2.
[18] Carrol ED, Campbell ME, Shaw BN, Pilling DW. Congenital lobar emphysema in congenital cytomegalovirus infection. Pediatr Radiol 1996;26:900–2.
[19] Kasales CJ, Coulson CC, Meilstrup JW, Ambrose A, Botti JJ, Holley GP. Diagnosis and differentiation of congenital diaphragmatic hernia from other noncardiac thoracic fetal masses. Am J Perinatol 1998;15:623–62.
[20] Daltro P, Werner H, Gasparetto TD, Domingues RC, Rodrigues L, Marchiori E, et al. Congenital chest malformations: a multimodality approach with emphasis on fetal MR imaging. Radiographics 2010;30:385–95.
[21] Liu YP, Lin YL, Chen SC. Fetal magnetic resonance imaging of congenital chest malformations: a pictorial review. J Radiol Sci 2013;38:119–27.
[22] Levine D, Barnewolt CE, Mehta TS, Trop I, Estroff J, Wong G. Fetal thoracic abnormalities: MR imaging. Radiology 2003;228:379–88.
[23] Pacharn P, Kline-Fath B, Calvo-Garcia M, Linam LE, Rubio EI, Salisbury S, et al. Congenital lung lesions: prenatal MRI and postnatal findings. Pediatr Radiol 2013;43:1136–43.
[24] Recio Rodríguez M, Martínez de Vega V, Cano Alonso R, Carrascoso Arranz J, Martínez Ten P, Pérez Pedregosa J. MR imaging of thoracic abnormalities in the fetus. Radiographics 2012;32:E305–21.
[25] Truitt AK, Carr SR, Cassese J, Kurkchubasche AG, Tracy Jr TF, Luks Fl. Perinatal management of congenital cystic lung lesions in the age of minimally invasive surgery. J Pediatr Surg 2006;41:893–6.
[26] Kunisaki SM, Fauza DO, Nemes LP, Barnewolt CE, Estroff JA, Kozakewich HP, et al. Bronchial atresia: the hidden pathology within a spectrum of prenatally diagnosed lung masses. J Pediatr Surg 2006;41:61–5.
[27] Kohl T, Gembruch U, Tchatcheva K, Schaible T. Two cases of prenatally diagnosed congenital lobar emphysema caused by lobar bronchial atresia. J Pediatr Surg 2006;41:E17–20.
[28] Doull IJ, Connett GJ, Warner JO. Bronchoscopic appearances of congenital lobar emphysema. Pediatr Pulmonol 1996;21:195–7.
[29] Thompson J, Forfar JO. Regional obstructive emphysema in infancy. Arch Dis Child 1958;33:97–101.
[30] Hendren WH, McKee DM. Lobar emphysema of infancy. J Pediatr Surg 1996;1:24–39.
[31] Stigers KB, Woodring JH, Kanga JF. The clinical and imaging spectrum of findings in patients with congenital lobar emphysema. Pediatr Pulmonol 1992;14:160–70.
[32] Donnelly LF, Frush DP. Localized radiolucent chest lesions in neonates: causes and differentiation. AJR Am J Roentgenol 1999;172:1651–8.
[33] Stanger P, Lucas Jr RL, Edwards JE. Anatomic factors causing respiratory distress in acyanotic congenital cardiac disease. Special reference to bronchial obstruction. Pediatrics 1969;43:760–9.
[34] Lincoln JC, Stark J, Subramanian S, Aberdeen E, Bonham-Carter RE, Berry CL, et al. Congenital lobar emphysema. Ann Surg 1971;173:55–62.
[35] Stocker JT. Cystic lung disease in infants and children. Fetal Pediatr Pathol 2009;28:155–84.
[36] Grethel EJ, Wagner AJ, Clifton MS, Cortes RA, Farmer DL, Harrison MR, et al. Fetal intervention for mass lesions and hydrops improves outcome: a 15-year experience. J Pediatr Surg 2007;42:117–23.
[37] Stocker JT, Madewell JE, Drake RM. Congenital cystic adenomatoid malformation of the lung. Classification and morphologic spectrum. Hum Pathol 1977;8:155–71.
[38] Adzick NS, Harrison MR, Glick PL, Golbus MS, Anderson RL, Mahony BS, et al. Fetal cystic adenomatoid malformation: prenatal diagnosis and natural history. J Pediatr Surg 1985;20:483–8.
[39] Beydon N, Larroquet M, Coulomb A, Jouannic JM, Ducou le Pointe H, Clément A, et al. Comparison between US and MRI in the prenatal assessment of lung malformations. Pediatr Radiol 2013;43:685–96.
[40] Shinmoto H, Kashima K, Yuasa Y, Tanimoto A, Morikawa Y, Ishimoto H, et al. MR imaging of non-CNS fetal abnormalities: a pictorial essay. Radiographics 2000;20:1227–43.
[41] Chen CP, Liu YP, Lin SP, Sheu JC, Hsu CY, Chang TY, et al. Prenatal magnetic resonance imaging demonstration of the systemic feeding artery of a pulmonary sequestration associated with in utero regression. Prenat Diagn 2005;25:721–3.
[42] Savic B, Birtel FJ, Tholen W, Funke HD, Knoche R. Lung sequestration: report of seven cases and review of 540 published cases. Thorax 1979;34:96–101.
[43] Azizkhan RG, Crombleholme TM. Congenital cystic lung disease: contemporary antenatal and postnatal management. Pediatr Surg Int 2008;24:643–57.
[44] Zeidan S, Hery G, Lacroix F, Gorincour G, Potier A, Dubus JC, et al. Intralobar sequestration associated with cystic adenomatoid malformation: diagnostic and thoracoscopic pitfalls. Surg Endosc 2009;23:1750–3.
[45] Cavoretto P, Molina F, Poggi S, Davenport M, Nicolaides KH. Prenatal diagnosis and outcome of echogenic fetal lung lesions. Ultrasound Obstet Gynecol 2008;32:769–83.
[46] Schwartz MZ, Ramachandran P. Congenital malformations of the lung and mediastinum-a quarter century of experience from a single institution. J Pediatr Surg 1997;32:44–7.
[47] Bailey PV, Tracy Jr T, Connors RH, deMello D, Lewis JE, Weber TR. Congenital bronchopulmonary malformations. J Thorac Cardiovasc Surg 1990;99:597–602. discussion 602–3.
[48] Roggin KK, Breuer CK, Carr SR, Hansen K, Kurkchubasche AG, Wesselhoeft Jr CW, et al. The unpredictable character of congenital cystic lung lesions. J Pediatr Surg 2000;35:801–5.
[49] Butterworth SA, Blair GK. Postnatal spontaneous resolution of congenital cystic adenomatoid malformations. J Pediatr Surg 2005;40:832–4.
[50] Calvert JK, Boyd PA, Chamberlain PC, Said S, Lakho K. Outcome of antenatally suspected congenital cystic adenomatoid malformation of the lung: 10 years' experience 1991–2001. Arch Dis Child Fetal Neonatal Ed 2006;91:26–8.